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Passive smoking and an increased risk of acute stroke

EDITOR,—Although "passive smoking" may be intuitively harmful, the paper by Bonita and colleagues[1] on the risk of stroke and environmental tobacco smoke (ETS) exposure suffers from two fundamental defects. The first is the enormously disproportionate effect due to a small exposure, and the second is the lack of allowance for confounding variables, especially diet.

Serum cotinine concentrations have recently been determined at the US National Center for Environmental Health using the most sensitive method to date of high resolution gas chromatography with mass spectrometry.[2] In 10 000 subjects it was shown that the mean serum cotinine concentration in ETS exposed non-smokers was 0.6 ng/ml compared to 300 ng/ml in active smokers. This represents 1/500th of the dose received by the active smoker.

It is difficult to reconcile this degree of exposure with an increased risk of stroke which is one quarter that of the active smoker. A similar disproportionate effect has been claimed for the increased risk of ischaemic heart disease and ETS exposure, but the biological plausibility and mechanisms of effect advanced to support this have been shown to lack credibility.[3 4]

It is well established that active smokers have other associated risk factors. They are physically less active and have lower intakes of fruit, vegetables, folate, and flavenoids,[5] which are all linked to a substantial increased risk for stroke,[6–8] and many of these characteristics are shared with non-smokers living with smokers.[9]

Although Bonita and colleagues excluded Maori and Pacific islands people from the study, the fact remains that in the residual sample, smoking, and therefore passive smoking, is more prevalent among lower socioeconomic groups, and independent of smoking, these groups have a higher risk of stroke.

The Pacific islands people indigenous to New Zealand have a higher incidence of stroke than Europeans indigenous to New Zealand. In this respect it is noteworthy that in the Pacific Melanesian islands where a traditional way of life is followed, but where cigarette smoking is excessive, cardiovascular disease and stroke are apparently absent. An example is the study on the Kitavan islanders, where 80% of people smoke cigarettes rolled from black imported or home grown tobacco and stroke is absent. Bonita and Beaglehole[10] in their comment on this study noted " . . .this is worrisome in view of the other adverse effects of tobacco". The staple diet of these people consists of root tubers, fruit, fish and coconuts, low salt, low fat (rather different to the New Zealand diet), they are physically active, and have low body mass index.

High stroke rates in Japan have diminished in recent years, due not to smoking reduction, but largely to salt restriction and a more westernised diet; the high stroke incidence in China is not strongly associated with smoking.

The interaction of diet, ethnicity, socioeconomic, cultural, and behavioural characteristics is complex, but cannot be ignored when considering the effect of smoking on the incidence of stroke. In view of the extremely low exposure and lack of allowance for confounding variables, the increased risk of stroke attributed to passive smoking by Bonita and colleagues[1] is unlikely to be true.

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Response by authors:

Passive smoking and risk of stroke seems a solid connection

EDITOR,—Kenneth Denson refers to results from the US National Center for Environmental Health where the serum cotinine concentration in environmental tobacco smoke (ETS) exposed non-smokers was only 1/500th of the dose received by the active smoker. From this point of view, Denson finds it difficult to reconcile that ETS exposed non-smokers in our study should have a risk of stroke one quarter that of the active smoker.

Although cotinine is a marker of tobacco smoke exposure, with its own limitations,[1] it has not been proved also to be a valid marker of a person's exposure to all of the toxic compounds in tobacco smoke. There are several possible biological mechanisms by which passive smoking may increase the risk of stroke—for example, increased platelet aggregation[2] and reduced oxygen carrying capacity.[3] Debate continues as to the best biomarker for passive smoking.

While it is true that the National Health and Nutrition Examination Survey (NHANES) study[4] cited by Denson was based on a large and carefully selected sample, it is noteworthy that the physical examinations and collection of blood sample "usually occurred 2 to 3 weeks after a household interview", and, furthermore, after the topic of smoking had already been raised. Thus, there was ample opportunity for members of each selected household to change their smoking behaviour well before the blood samples were drawn. Cotinine concentrations would then not have been indicative of usual patterns of exposure to ETS. In addition, NHANES assumed that sharing a home with a smoker equated with passive exposure. This assumption becomes particularly tenuous when 40% of participants in the study were aged less than 12 years; the effects of passive smoking on the health of children were already well known in the community.

While it would have been optimal to have been able to control for differences in diet between non-smokers exposed and not exposed to ETS, confounding is unlikely to explain our findings. There is only limited evidence that the diet of individuals strongly affects their risk of stroke. In general terms, the relative risk associated with a confounding variable needs to be at least double the observed association for that confounder to explain it. Denson is unable to nominate a specific confounder and refers instead to ecological studies which are well known as having many pitfalls. It is highly unlikely that decades of work on the aetiology of stroke, including a number of very large prospective studies, would have failed to uncover a strong dietary risk factor for stroke, if one existed. In the meta-analysis of analytical studies by Law and colleagues[2] differences in diet were judged likely to account for 6% of the increased risk of coronary heart disease associated with ETS in non-smokers. If those results may be extrapolated to our data on stroke the odds ratio would decrease to 1.72 (1.82/1.06)—which is still a considerable increased risk. Thus, dietary differences are unlikely to explain all of the increased risk in non-smokers exposed to ETS in the present study.[5]

It is always a possibility that one study, by chance, finds a strong association between an exposure and an end point. What accounts to the credibility of our study is that the anti-tobacco campaign in New Zealand has been very successful. In the study of environmental tobacco smoking exposure in the US population, the authors found that 88% of people who were not smokers had detectable concentrations of cotinine, including people who reported not to be exposed either at home or at work.[4] Thus, the relatively high odds ratios found in our

study, for active as well as passive smoking, could simply reflect a satisfactory allocation of non-smokers not exposed to passive smoking.

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A tentative illustration of the smoking initiation and cessation cycles

EDITOR,—In many former papers,[1–4] the smoker's career is described separately for the processes of initiation and cessation. Furthermore, the recently advocated issues of smoking reduction, sometimes followed by a secondary cessation[5–7] are not always considered. We have tried to summarise the complete smoker's career in one single schema (fig 1) in a way that could be useful for teaching purposes in the preventive and curative fields.

The non-smoker (A), after a preparatory stage, becomes an occasional smoker (B) (trying and experimentation stages) and afterwards, exceptionally abandons smoking. In most cases, however, experimental smokers progress toward regular, daily use (C). The stage labelled "happy smoker" (D) usually lasts for many years, after which smokers perceive more acutely the "pros" and "cons" of their tobacco use, thus becoming "ambivalent smokers" (E). Later on, some prepare to stop (F), and start to take action (primary cessation) (G), which is sometimes followed by perseverance (H). In most cases, because of withdrawal symptoms, cessation is followed by a relapse (I) and the smoker progresses further, often several times, into the cessation cycle through the stages of "ambivalence" and "readiness to stop" before finally succeeding with cessation and becoming a persistent "happy ex-smoker" (H). Some smokers are unable to quit completely but can space their smoking, again becoming occasional smokers (sometimes by using pipes, cigars or cigarillos instead of cigarettes), while others reduce their daily cigarette consumption, often nowadays with the help of concomitant nicotine substitution, in a process of "harm reduction". Some of these smokers finally quit (secondary cessation) (J) to also become "happy ex-smokers". However some remain continuing smokers (K) until their death.

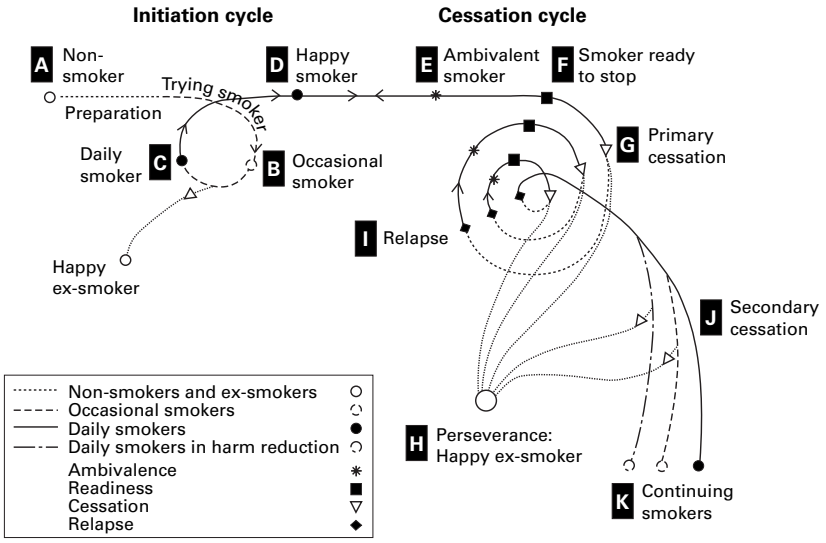

Figure 1 Diagram summarising the complete smoker's career from initiation to cessation.

In most cases, the process evolves in the described direction, but, as recently stressed by Butler and colleagues,[8] unfortunate interventions, especially at the stage of ambivalence, can induce a regression in the cessation cycle and delay quitting by reinforcing the smoker's resistance to change.

Personal variables largely influence the speed of movement through both the initiation and cessation cycles, while external interventions as well as emerging anti-smoking social norms are conducive to change.

A universally applicable quantitative assessment of the mean durations of the various stages is not feasible, since they differ according to different settings. Similarly, the distribution of the population of smokers in the various stages also differs according to national, ethnic, and socioeconomical parameters.

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